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Effect of dendritic backpropagating action potential on neural interaction

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Abstract

We elucidate the effect of dendritic backpropagating action potentials on neural interactions. We propose a neural oscillator model that mimics the backpropagating potentials upon the dendrite. We show that backpropagating potentials change the stability structure of the system from monostable to bistable. Such bistability causes the multiple phase clustering of the neural population. The phase clustering is a cogent hypothesis to explain the information representation binding distributed codes in the brain.

Keywords: Dendrite; Backpropagation; Synaptic response; Oscillator

It has been generally recognized that electrical signals within a nerve cell flow in a *feed-forward* direction: from the input sites (usually the dendrite) to the output sites (usually the soma). This hypothesis is referred to as the principle of dynamic polarization [8]. Almost all formal neuron models in theoretical studies are based

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upon this hypothesis. In such formal neuron models, the dendrite plays a role of just a cable transmitting synaptic input signals to the soma. However, the properties of the dendrite are more complicated than those of the formal neuron model. It has been reported that many kinds of voltage-activated ion channels are highly distributed over the dendrite and that action potentials can be generated in the dendrites [9]. Under some conditions, action potentials are initiated at the dendrite by strong EPSP and propagate to the soma (forward propagation). Under some conditions, action potentials are initialized at the soma even by strong EPSP and propagate to the dendritic input sites (backpropagation). In this paper, we focus on the role of the backpropagation in the information processing of a single nerve cell. Backpropagation implies that output signals are fed back to the input sites. There is a possibility that in this case, a single nerve cell might be able to acquire a capacity of *active* information processing through the modification of synaptic responses by backpropagating action potentials. There are many reports on the relationship between backpropagating action potential and postsynaptic potential [3,4,7,10]. All these reports have focused on the amplification of backpropagating action potential and shunting inhibition of postsynaptic potential that are caused by interference between these two potentials. If the temporal coding hypothesis (that the information is coded in spike timing) is accepted, any discussion on neural information processing should focus on the timing modification of action potential initiation by the backpropagation rather than the amplification/inhibition of these potentials.

We analyze the impact of the spike-timing modification by backpropagation on neural interaction and neural cooperative behavior. We measure in regular oscillatory states the phase modification of action potential initiation as a function of relative timing between action potential initiation and postsynaptic potential generation. We propose a neural oscillator model that mimics backpropagating potentials upon the dendrite. This model enables us to extract the effect of the backpropagating potential only. Through the phase reduction, we demonstrate that at the synapse, a collision of synaptic inputs with backpropagating potentials induces bistable states consisting of in-phase locking and anti-phase locking. Models previously proposed [1,11] are similar to our model proposed here, but those authors did not clearly mention the role of the backpropagation. In the previous studies, the phase description of whole systems has been numerically derived. The numerical calculation incorporated and renormalized the effects of the backpropagation into only a *phase-coupling function* to establish the phase equation, thus the effects of the backpropagation could not be dissociated from the phase equation. On the other hand, in our analysis, we clearly separate the effects of the backpropagation from the phase equation. In the phase equation obtained here, the dynamics of dendrite and synapse are represented only by the *transfer function* or *describing function* that describes their frequency response. Our theoretical result that a collision of synaptic inputs with backpropagating potentials causes such bistable states is a concrete example that validates physiological conjectures regarding the function of back-propagations. Through our analytical phase reduction we can elucidate the influence that the transmission lag caused by the dendrite and the synapse, which depends on